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Search Results - Record(s) 1 through 10 of 59 returned.

☐ 1. Document ID: US 20050089844 A1

L3: Entry 1 of 59

File: PGPB

Apr 28, 2005

PGPUB-DOCUMENT-NUMBER: 20050089844

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20050089844 A1

TITLE: Novel dual oxidases as mitogenic and endocrine regulators

PUBLICATION-DATE: April 28, 2005

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Lambeth, J. David	Decatur	GA	US	
Lassegue, Bernard P.	Decatur	GA	US	
Griendling, Kathy K.	Stone Mountain	GA	US	
Arnold, Rebecca S.	Lilburn	GA	US	
Cheng, Guangjie	Atlanta	GA	US	
Sharling, Lisa	Scotland	GA	GB	
Benian, Guy	Decatur	GA	US	
Edens, William A.	Tucker		US	

US-CL-CURRENT: [435/6](#); [435/189](#), [435/320.1](#), [435/325](#), [435/69.1](#), [536/23.2](#)

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KMC	Draw D
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☐ 2. Document ID: US 20050003412 A1

L3: Entry 2 of 59

File: PGPB

Jan 6, 2005

PGPUB-DOCUMENT-NUMBER: 20050003412

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20050003412 A1

TITLE: Mitogenic oxygenase regulators

PUBLICATION-DATE: January 6, 2005

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Lambeth, J. David	Atlanta	GA	US	

Cheng, Guangjie Doraville GA US

US-CL-CURRENT: 435/6; 435/192, 435/320.1, 435/325, 435/69.1, 536/23.2

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KMC	Draw. De
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☐ 3. Document ID: US 20040253681 A1

L3: Entry 3 of 59

File: PGPB

Dec 16, 2004

PGPUB-DOCUMENT-NUMBER: 20040253681

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20040253681 A1

TITLE: Mitogenic oxygenase regulators

PUBLICATION-DATE: December 16, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Lambeth, J. David	Atlanta	GA	US	
Cheng, Guangjie	Doraville	GA	US	

US-CL-CURRENT: 435/69.1; 435/189, 435/320.1, 435/325, 536/23.2

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KMC	Draw. De
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☐ 4. Document ID: US 20040148645 A1

L3: Entry 4 of 59

File: PGPB

Jul 29, 2004

PGPUB-DOCUMENT-NUMBER: 20040148645

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20040148645 A1

TITLE: ATP diphosphohydrolase (CD39) gene therapy for inflammatory or thrombotic conditions and transplantation and means therefor

PUBLICATION-DATE: July 29, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Bach, Fritz H.	Boston	MA	US	
Robson, Simon	Brookline	MA	US	
Beaudoin, Adrien R.	Rock Forest	MA	CA	
Sevigny, Jean	Brookline		US	

US-CL-CURRENT: 800/8; 424/93.21

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KMC	Draw. De
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☐ 5. Document ID: US 20040142391 A1

L3: Entry 5 of 59

File: PGPB

Jul 22, 2004

PGPUB-DOCUMENT-NUMBER: 20040142391

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20040142391 A1

TITLE: Methods for determining whether a compound is capable of inhibiting the interaction of a peptide with RAGE

PUBLICATION-DATE: July 22, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Schmidt, Ann Marie	Franklin Lakes	NJ	US	
Stern, David	Great Neck	NY	US	

US-CL-CURRENT: 435/7.2

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KMC	Draw D
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☐ 6. Document ID: US 20040109875 A1

L3: Entry 6 of 59

File: PGPB

Jun 10, 2004

PGPUB-DOCUMENT-NUMBER: 20040109875

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20040109875 A1

TITLE: Pro-apoptotic bacterial vaccines to enhance cellular immune responses

PUBLICATION-DATE: June 10, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Kernodle, Douglas S.	Brentwood	TN	US	
Bochan, Markian R	Nashville	TN	US	

US-CL-CURRENT: 424/200.1; 435/252.3

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KMC	Draw D
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☐ 7. Document ID: US 20040093628 A1

L3: Entry 7 of 59

File: PGPB

May 13, 2004

PGPUB-DOCUMENT-NUMBER: 20040093628

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20040093628 A1

TITLE: Methods and transgenic mouse model for identifying and modulating factors involved in the production of reactive oxygen intermediates

PUBLICATION-DATE: May 13, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Lambeth, J. David	Atlanta	GA	US	
Cheng, Guangjie	Atlanta	GA	US	
McCoy, James	Atlanta	GA	US	

US-CL-CURRENT: 800/18; 435/354

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KMC	Draw D
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☐ 8. Document ID: US 20040091466 A1

L3: Entry 8 of 59

File: PGPB

May 13, 2004

PGPUB-DOCUMENT-NUMBER: 20040091466

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20040091466 A1

TITLE: Regulatory protein for nox enzymes

PUBLICATION-DATE: May 13, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Lambeth, J. David	Atlanta	GA	US	
Cheng, Guangjie	Atlanta	GA	US	

US-CL-CURRENT: 424/94.4; 435/189

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KMC	Draw D
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☐ 9. Document ID: US 20040043934 A1

L3: Entry 9 of 59

File: PGPB

Mar 4, 2004

PGPUB-DOCUMENT-NUMBER: 20040043934

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20040043934 A1

TITLE: Synthetic peptides that inhibit leukocyte superoxide anion production and/or attract leukocytes

PUBLICATION-DATE: March 4, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Blecha, Frank	Manhattan	KS	US	
Shi, Jishu	Los Angeles	CA	US	

US-CL-CURRENT: 514/12

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KWIC	Draw De
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☐ 10. Document ID: US 20040038222 A1

L3: Entry 10 of 59

File: PGPB

Feb 26, 2004

PGPUB-DOCUMENT-NUMBER: 20040038222

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20040038222 A1

TITLE: Anthrax susceptibility gene

PUBLICATION-DATE: February 26, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Dietrich, William F.	Newton	MA	US	
Watters, James W.	St. Louis	MO	US	

US-CL-CURRENT: 435/6; 435/194, 435/320.1, 435/325, 435/69.1, 536/23.2

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KWIC	Draw De
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L2 and reactive oxygen intermediate?

59

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<input type="checkbox"/>	L3	L2 and reactive oxygen intermediate?	59
<input type="checkbox"/>	L2	L1 and human	1535
<input type="checkbox"/>	L1	(NADPH oxidase or NOX)	43473

END OF SEARCH HISTORY

=> d 13 1-10 ibib ab

L3 ANSWER 1 OF 10 MEDLINE on STN
ACCESSION NUMBER: 2004281392 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15181570
TITLE: **DNA** phasing by TA dinucleotide microsatellite
length determines in vitro and in vivo expression of the
gp91phox subunit of **NADPH oxidase** and
mediates protection against severe malaria.
AUTHOR: Uhlemann Anne-Catrin; Szlezak Nicole A; Vonthein Reinhard;
Tomiuk Jurgen; Emmer Stefanie A; Lell Bertrand; Kremsner
Peter G; Kun Jurgen F J
CORPORATE SOURCE: Department of Parasitology, Institute of Tropical Medicine,
University of Tübingen, Tübingen, Germany..
a.uhlemann@sghms.ac.uk
SOURCE: Journal of infectious diseases, (2004 Jun 15) 189 (12)
2227-34. Electronic Publication: 2004-05-25.
Journal code: 0413675. ISSN: 0022-1899.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200407
ENTRY DATE: Entered STN: 20040608
Last Updated on STN: 20040709
Entered Medline: 20040708

AB **Reactive oxygen intermediates** (ROIs) play a
major role in the nonspecific innate immune response to invading
microorganisms, such as *Plasmodium falciparum*. In a search for genetic
markers that determine differences in production of ROI, we detected a
highly polymorphic region of dinucleotide TA repeats approximately 550 bp
upstream of the **NADPH oxidase** gp91(phox) subunit
promoter. We genotyped 183 matched Gabonese children with severe or mild
malaria. Repeat lengths TA(11) and TA(16) differed significantly in
frequency between mild and severe infection, which suggests protection
against severe malaria. Both repeat lengths showed lower levels of
NADPH oxidase and promoter activities, which can be
explained by a cyclic trend in TA repeat length with a period of
approximately 5, which indicates the necessity of correct **DNA**
phasing between 2 possible control regions in the promoter. We provide a
molecular model of how **DNA** phasing generated by TA dinucleotide
polymorphisms may influence the expression level and protect against
severe malaria.

L3 ANSWER 2 OF 10 MEDLINE on STN
ACCESSION NUMBER: 2002130980 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11859135
TITLE: Susceptibility of IFN regulatory factor-1 and IFN consensus
sequence binding protein-deficient mice to brucellosis.
AUTHOR: Ko Jinkyung; Gendron-Fitzpatrick Annette; Splitter Gary A
CORPORATE SOURCE: Laboratory of Cellular and Molecular Immunology, Department
of Animal Health and Biomedical Sciences, University of
Wisconsin, Madison, WI 53706, USA.
CONTRACT NUMBER: R01 AI 48490 (NIAID)
SOURCE: Journal of immunology (Baltimore, Md. : 1950), (2002 Mar 1)
168 (5) 2433-40.
Journal code: 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200203
ENTRY DATE: Entered STN: 20020228
Last Updated on STN: 20020317
Entered Medline: 20020315

AB IFN-gamma is a key cytokine controlling *Brucella* infection, and the
diverse functions of this cytokine are mediated by IFN regulatory factors
(IRFs) such as IRF-1, IRF-2, and IFN consensus sequence binding protein
(ICSBP). However, the roles of these three IRFs in *Brucella* infection

have not been investigated. The infection of each IRF-deficient mouse strain provides an opportunity to determine not only the significance of each IRF molecule but also the crucial immune components necessary for host defense during in vivo infection, because respective IRF-deficient mouse strains contain unique immunodeficient phenotypes. *Brucella abortus* S2308-infected IRF-1-/- mice were dead within 2 wk postinfection, while IRF-2-/- mice contained less splenic *Brucella* CFU than wild-type mice at the early stage of infection. Infected ICSBP-/- mice maintained a plateau of splenic *Brucella* CFU throughout the infection. Additional infection of IL-12p40-, NO synthase 2-, and gp91(phox)-deficient mice indicates that these immune components are crucial for *Brucella* immunity and may contribute to the susceptibility of IRF-1-/- and ICSBP-/- mice. Immunologic and histopathological analyses of infected IRF-1-/- mice indicate that the absence of IL-12p40 induction and serious hepatic damage are involved in the death of IRF-1-/- mice. These results indicate that 1) IRF-1 and ICSBP are essential transcriptional factors for IFN-gamma-mediated protection against *Brucella*; 2) IL-12, reactive nitrogen intermediates, and **reactive oxygen intermediates** are crucial immune components against *Brucella*, and their absence may contribute to the susceptibility of IRF-1-/- and ICSBP-/- mice; and 3) hepatic damage caused by *Brucella* virulence contributes to the death of IRF-1-/- mice.

L3 ANSWER 3 OF 10 MEDLINE on STN
 ACCESSION NUMBER: 2001527204 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 11461902
 TITLE: lalpha,25-Dihydroxyvitamin D3-induced monocyte antimycobacterial activity is regulated by phosphatidylinositol 3-kinase and mediated by the NADPH-dependent phagocyte oxidase.
 AUTHOR: Sly L M; Lopez M; Nauseef W M; Reiner N E
 CORPORATE SOURCE: Department of Medicine (Division of Infectious Diseases), University of British Columbia, Faculties of Medicine and Science, Research Institute of the Vancouver Hospital and Health Sciences Center, Vancouver, British Columbia V5Z 3J5, Canada.
 CONTRACT NUMBER: AI-34879 (NIAID)
 NO1-AI-75320 (NIAID)
 SOURCE: Journal of biological chemistry, (2001 Sep 21) 276 (38) 35482-93. Electronic Publication: 2001-07-18.
 Journal code: 2985121R. ISSN: 0021-9258.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200110
 ENTRY DATE: Entered STN: 20011001
 Last Updated on STN: 20030105
 Entered Medline: 20011025
 AB We investigated the basis for the induction of monocyte antimycobacterial activity by lalpha,25-dihydroxyvitamin D(3) (D(3)). As expected, incubation of *Mycobacterium tuberculosis*-infected THP-1 cells or **human** peripheral blood, monocyte-derived macrophages with hormone resulted in the induction of antimycobacterial activity. This effect was significantly abrogated by pretreatment of cells with either of the phosphatidylinositol 3-kinase (PI 3-K) inhibitors, wortmannin or LY294002, or with antisense oligonucleotides to the p110 subunit of PI 3-K. Cells infected with *M. tuberculosis* alone or incubated with D(3) alone produced little or undetectable amounts of superoxide anion (O(2)). In contrast, exposure of *M. tuberculosis*-infected cells to D(3) led to significant production of O(2), and this response was eliminated by either wortmannin, LY294002, or p110 antisense oligonucleotides. As was observed for PI 3-K inactivation, the **reactive oxygen intermediate** scavenger, 4-hydroxy-TEMPO, and degradative enzymes, polyethylene glycol coupled to either superoxide dismutase or catalase, also abrogated D(3)-induced antimycobacterial activity. Superoxide production by THP-1 cells in response to D(3) required prior infection with live *M. tuberculosis*, since exposure of cells to either killed *M. tuberculosis* or latex beads did not prime for an oxidative burst in

response to subsequent hormone treatment. Consistent with these findings, redistribution of the cytosolic oxidase components p47(phox) and p67(phox) to the membrane fraction was observed in cells incubated with live M. tuberculosis and D(3) but not in response to combined treatment with heat-killed M. tuberculosis followed by D(3). Redistribution of p47(phox) and p67(phox) to the membrane fraction in response to live M. tuberculosis and D(3) was also abrogated under conditions where PI 3-K was inactivated. Taken together, these results indicate that D(3)-induced, **human** monocyte antimycobacterial activity is regulated by PI 3-K and mediated by the NADPH-dependent phagocyte oxidase.

L3 ANSWER 4 OF 10 MEDLINE on STN
ACCESSION NUMBER: 1999054991 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9835621
TITLE: Role of oxygen radicals generated by **NADPH oxidase** in apoptosis induced in **human** leukemia cells.
AUTHOR: Hiraoka W; Vazquez N; Nieves-Neira W; Chanock S J; Pommier Y
CORPORATE SOURCE: Laboratory of Molecular Pharmacology, National Cancer Institute, Bethesda, Maryland 20892, USA.
SOURCE: Journal of clinical investigation, (1998 Dec 1) 102 (11) 1961-8.
Journal code: 7802877. ISSN: 0021-9738.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199901
ENTRY DATE: Entered STN: 19990128
Last Updated on STN: 19990128
Entered Medline: 19990108

AB We have used a **human** leukemia cell line that, after homologous recombination knockout of the gp91-phox subunit of the phagocyte respiratory-burst oxidase cytochrome b-558, mimics chronic granulomatous disease (X-CGD) to study the role of oxygen radicals in apoptosis. Camptothecin (CPT), a topoisomerase I inhibitor, induced significantly more apoptosis in PLB-985 cells than in X-CGD cells. Sensitivity to CPT was enhanced after neutrophilic differentiation, but was lost after monocytic differentiation. No difference between the two cell lines was observed after treatment with other apoptosis inducers, including etoposide, ultraviolet radiation, ionizing radiation, hydrogen peroxide, or 7-hydroxystaurosporine. After granulocytic differentiation of both cell lines, CPT still induced apoptosis, suggesting independence from replication in fully differentiated and growth-arrested cells. Pyrrolidine dithiocarbamate (an antioxidant inhibitor of NF-kappaB) and catalase partially inhibited CPT-induced **DNA** fragmentation in granulocytic-differentiated PLB-985 cells, but had no effect in X-CGD cells. Flow cytometry analysis revealed that **reactive oxygen intermediates** were generated in CPT-treated PLB-985 cells. These data indicate that oxygen radicals generated by **NADPH oxidase** may contribute directly or indirectly to CPT-induced apoptosis in **human** leukemia and in neutrophilic-differentiated cells.

L3 ANSWER 5 OF 10 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2002:793812 HCAPLUS
DOCUMENT NUMBER: 137:305746
TITLE: Protein and cDNA sequences of **human** mitogenic oxygenase regulators: **Nox 4** and **Nox 5** and therapeutic uses thereof
INVENTOR(S): Lambeth, J. David; Cheng, Guangjie
PATENT ASSIGNEE(S): Emory University, USA
SOURCE: PCT Int. Appl., 91 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002081703	A2	20021017	WO 2001-US51495	20011115
WO 2002081703	A3	20031224		
WO 2002081703	C2	20040415		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2429376	AA	20021017	CA 2001-2429376	20011115
US 2002176852	A1	20021128	US 2001-999248	20011115
US 6846672	B2	20050125		
EP 1399565	A2	20040324	EP 2001-273646	20011115
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2004537981	T2	20041224	JP 2002-580066	20011115
US 2004253681	A1	20041216	US 2004-850060	20040520
US 2005003412	A1	20050106	US 2004-850103	20040520

PRIORITY APPLN. INFO.:

US 2000-249305P	P	20001116
US 2000-251364P	P	20001205
US 2001-289172P	P	20010507
US 2001-289537P	P	20010507
US 2001-999248	A1	20011115
WO 2001-US51495	W	20011115

AB The present invention relates to protein and cDNA sequences of new gene of **human** mitogenic oxygenase regulators: **Nox 4** and **Nox 5** involved in generation of **reactive oxygen intermediates** that affect cell division. The present invention also provides vectors contg. these genes, cells transfected with these vectors, antibodies raised against these novel proteins, kits for detection, localization and measurement of these genes and proteins, and methods to det. the activity of drugs to affect the activity of the proteins of the present invention.

L3 ANSWER 6 OF 10 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2001:851225 HCAPLUS

DOCUMENT NUMBER: 136:2247

TITLE: **Human** and nematode dual oxidases as mitogenic and endocrine regulators

INVENTOR(S): Lambeth, J. David; Lassegue, Bernard P.; Griendling, Kathy K.; Arnold, Rebecca S.; Cheng, Guangjie; Sharling, Lisa; Benian, Guy; Edens, William A.

PATENT ASSIGNEE(S): Emory University, USA

SOURCE: PCT Int. Appl., 76 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001087957	A2	20011122	WO 2001-US15573	20010514
WO 2001087957	C2	20021212		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,				

BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 6620603 B1 20030916 US 1999-437568 19991110
CA 2409068 AA 20011122 CA 2001-2409068 20010514
EP 1285071 A2 20030226 EP 2001-939033 20010514
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
JP 2003533217 T2 20031111 JP 2001-585175 20010514
US 2005089844 A1 20050428 US 2002-276153 20010514
US 2003157678 A1 20030821 US 2002-318906 20021213
US 2003166198 A1 20030904 US 2002-319236 20021213

PRIORITY APPLN. INFO.: US 1999-437568 A2 19991110
US 2000-204441P P 20000515
US 2000-222421P P 20000801
US 1998-107911P P 19981110
US 1999-149332P P 19990817
US 1999-151242P P 19990827
WO 2001-US15573 W 20010514

AB A novel family of nucleotide and sequences and encoded proteins, termed duox or "dual oxidase" proteins, are provided. The duox proteins have homol. to the gp91phox protein involved in **reactive oxygen intermediate** generation; however, the duox proteins comprise a novel and distinct family of proteins. The duox proteins comprise 3 distinct regions: the N-terminal regions has homol. to peroxidase proteins, the internal region has homol. to calmodulin proteins, and the C-terminal region has homol. to mox (also called **nox**) proteins. The cDNA nucleotide and amino acids sequences are provided for **human** duox2 and *Caenorhabditis elegans* duox1. Duox proteins are involved in generation of **reactive oxygen intermediates** and in peroxidative reactions that affect biol. functions including cell division, thyroid hormone biosynthesis, and tissue fibrosis. The present invention also provides vectors contg. these genes, cells transfected with these vectors, antibodies raised against these novel proteins, kits for detection, localization and measurement of these genes and proteins, and methods to det. the activity of drugs to affect the activity of the proteins of the present invention.

L3 ANSWER 7 OF 10 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1999:456576 HCAPLUS

DOCUMENT NUMBER: 131:225337

TITLE: Stimulation of a vascular smooth muscle cell NAD(P)H oxidase by thrombin. Evidence that p47phox may participate in forming this oxidase in vitro and in vivo

AUTHOR(S): Patterson, Cam; Ruef, Johannes; Madamanchi, Nageswara R.; Barry-Lane, Patricia; Hu, Zhaoyong; Horaist, Chris; Ballinger, Carol A.; Brasier, Alan R.; Bode, Christoph; Runge, Marschall S.

CORPORATE SOURCE: Division of Cardiology and Sealy Center for Molecular Cardiology, University of Texas Medical Branch, Galveston, TX, 77555-1064, USA

SOURCE: Journal of Biological Chemistry (1999), 274(28), 19814-19822

CODEN: JBCHA3; ISSN: 0021-9258

PUBLISHER: American Society for Biochemistry and Molecular Biology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Thrombin is a potent vascular smooth muscle cell (VSMC) mitogen. Because recent evidence implicates **reactive oxygen intermediates** (ROI) in VSMC proliferation in general and atherogenesis in particular, we investigated whether ROI generation is necessary for thrombin-induced mitogenesis. Treatment of **human** aortic smooth muscle cells with thrombin increased **DNA** synthesis, an effect that was antagonized by diphenyleneiodonium but not by other inhibitors of cellular oxidase systems. This effect of thrombin was accompanied by increased O₂.cntdot.- and H₂O₂ generation and NADH/NADPH consumption. ROI generation in response to thrombin pretreatment could also be blocked by diphenyleneiodonium, suggesting that the NAD(P)H oxidase was necessary for ROI generation and thrombin-induced

mitogenesis. Because of obsd. differences between the VSMC and neutrophil oxidase, we examd. whether the cytosolic components of the phagocytic NAD(P)H oxidase were present in VSMC. P47phox and Rac2 were present in VSMC. Furthermore, thrombin increased expression of p47phox and Rac2 and stimulated their translocation to the cell membrane. We examd. whether p47phox might be similarly regulated in vivo in a rat aorta balloon injury model and found that p47phox protein was increased after injury. Immunocytochem. localized expression of p47phox to the neointima and media of injured arteries. Our data demonstrate that generation of O2.cntdot.- and H2O2 is required for thrombin-mediated mitogenesis in VSMC and that p47phox is regulated by thrombin in vitro and is assocd. with vascular lesion formation in vivo.

REFERENCE COUNT: 63 THERE ARE 63 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 8 OF 10 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
 ACCESSION NUMBER: 2000:42162 BIOSIS
 DOCUMENT NUMBER: PREV200000042162
 TITLE: Role of the **NADPH oxidase** and **reactive oxygen intermediates** in hyperthermia induced apoptosis.
 AUTHOR(S): Pandrey, Joachim [Reprint author]; Schindler, Susann G.; Katschinski, Dorthé M.
 CORPORATE SOURCE: Institut fuer Physiologie, University Essen, Essen, Germany
 SOURCE: Blood, (Nov. 15, 1999) Vol. 94, No. 10 SUPPL. 1 PART 2, pp. 155b. print.
 Meeting Info.: Forty-first Annual Meeting of the American Society of Hematology. New Orleans, Louisiana, USA. December 3-7, 1999. The American Society of Hematology. CODEN: BLOOAW. ISSN: 0006-4971.
 DOCUMENT TYPE: Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 LANGUAGE: English
 ENTRY DATE: Entered STN: 26 Jan 2000
 Last Updated on STN: 31 Dec 2001

L3 ANSWER 9 OF 10 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
 ACCESSION NUMBER: 1996:11443 BIOSIS
 DOCUMENT NUMBER: PREV199698583578
 TITLE: Cytomegalovirus infection of **human** smooth muscle cells causes a prooxidant state that is mediated in part by **NADPH-oxidase**.
 AUTHOR(S): Speir, Edith; Shibutani, Tomoko; Yu, Zu-Xi; Epstein, Stephen E.
 CORPORATE SOURCE: National Inst. Health, Bethesda, MD, USA
 SOURCE: Circulation, (1995) Vol. 92, No. 8 SUPPL., pp. I230-I231.
 Meeting Info.: 68th Scientific Session of the American Heart Association. Anaheim, California, USA. November 13-16, 1995. CODEN: CIRCAZ. ISSN: 0009-7322.
 DOCUMENT TYPE: Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 LANGUAGE: English
 ENTRY DATE: Entered STN: 4 Jan 1996
 Last Updated on STN: 4 Jan 1996

L3 ANSWER 10 OF 10 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
 ACCESSION NUMBER: 1999076638 EMBASE
 TITLE: **Reactive oxygen intermediate** -dependent NF-.kappa.B activation by interleukin- 1.beta. requires 5-lipoxygenase or **NADPH oxidase** activity.
 AUTHOR: Bonizzi G.; Piette J.; Schoonbroodt S.; Greimers R.; Havard L.; Merville M.- P.; Bours V.
 CORPORATE SOURCE: V. Bours, Medical Oncology, CHU B35, Universite de Liege, 4000 Liege, Belgium. vbours@ulg.ac.be
 SOURCE: Molecular and Cellular Biology, (1999) Vol. 19, No. 3, pp. 1950-1960.

Refs: 67
ISSN: 0270-7306 CODEN: MCEBD4
United States
Journal; Article
022 Human Genetics
029 Clinical Biochemistry
English
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SUMMARY LANGUAGE:
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AB We previously reported that the role of **reactive oxygen intermediates** (ROIs) in NF- κ B activation by proinflammatory cytokines was cell specific. However, the sources for ROIs in various cell types are yet to be determined and might include 5-lipoxygenase (5-LOX) and **NADPH oxidase**. 5-LOX and 5-LOX activating protein (FLAP) are coexpressed in lymphoid cells but not in monocytic or epithelial cells. Stimulation of lymphoid cells with interleukin-1 β (IL-1 β) led to ROI production and NF- κ B activation, which could both be blocked by antioxidants or FLAP inhibitors, confirming that 5-LOX was the source of ROIs and was required for NF- κ B activation in these cells. IL-1 β stimulation of epithelial cells did not generate any ROIs and NF- κ B induction was not influenced by 5-LOX inhibitors. However, reintroduction of a functional 5-LOX system in these cells allowed ROI production and 5-LOX-dependent NF- κ B activation. In monocytic cells, IL-1 β treatment led to a production of ROIs which is independent of the 5-LOX enzyme but requires the **NADPH oxidase** activity. This pathway involves the Rac1 and Cdc42 GTPases, two enzymes which are not required for NF- κ B activation by IL-1 β in epithelial cells. In conclusion, three different cell-specific pathways lead to NF- κ B activation by IL-1 β : a pathway dependent on ROI production by 5-LOX in lymphoid cells, an ROI- and 5-LOX- independent pathway in epithelial cells, and a pathway requiring ROI production by **NADPH oxidase** in monocytic cells.

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FILE 'MEDLINE, HCAPLUS, BIOSIS, BIOTECHDS, SCISEARCH, EMBASE' ENTERED AT 10:17:05 ON 03 JUN 2005

L1 240 S REACTIVE OXYGEN INTERMEDIATE? AND HUMAN AND (NADPH OXIDASE O
L2 106 DUP REM L1 (134 DUPLICATES REMOVED)
L3 10 S L2 AND DNA

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